Mechanism Provoking Behavioral Changes in Patients with Chronic Toxoplasmosis Infections of the Brain May Be Related to Toxo Depletion of Esterases

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Introduction

While it is now understood that many cases of rage attacks may be attributable to neurological infection with toxoplasmosis and that this infection is, in most cases, traceable to contact with feline fecal matter combined with poor hand washing practices, the precise mechanisms through which toxoplasmosis adversely impacts neural function are poorly understood.

Abstract

Gait analysis of known toxoplasmosis patients indicates that the body language of an individual with toxoplasmosis tends inappropriately toward exaggerated swinging of the arms and over-pronounced stride. These functions of body language and gait are controlled by the motor cortex.

Although toxoplasmosis is associated primarily with behavioral changes, little attention has been paid to some of the more subtle motor-related changes the condition may bring about independent of any neural degradation. A partial depletion of chemicals in the esterase family could explain subtle changes in gait making the stride of a toxoplasmosis patient distinct from that of an uninfected individual as well as the behavioral tendency toward toxoplasmosis patients suffering from rage attacks.

This chemical depletion, brought about by toxoplasmosis' appetite for metabolizing esterases as they constitute a source of nourishment for the bacteria, results in a tendency for physical motions to extend beyond the intended maximal point. Examples of this sort of accidental over-extension from our everyday lives include, for instance, sliding a plate or cup on a counter and accidentally causing it to slide off the counter by using too much force. Individuals with a toxoplasmosis infection of the brain, if surveyed, would likely respond that they do this more frequently than most.

A deficit of esterase chemicals, which serve to keep other neurotransmitters in check, could also account for the inability of individuals suffering from rage attacks to calm down after experiencing an emotional insult. A human with optimally functional neurology will actually produce additional esterases to self-regulate rage once an individual becomes cognizant that, for example, their toe was accidentally stepped upon. The toxoplasmosis-affected patient, even after becoming conscious of the accidental nature of the insult cannot help but to continue experiencing that initial flash of rage over a longer-than-normal period

of time. Where most people might experience rage for an imperceptible fraction of a second or not at all from such a minor insult, an individual with a compromised esterase levels could be expected to remain enraged for 15 minutes or more each and every time even a slight insult is experienced. Although relief is eventually experienced, the length of time it takes for that relief to come about depends upon the extent of the compromise of the esterase chemistry.

Conclusion

If verified, this understanding could help to address many of the symptoms of toxoplasmosis as the research community continues to work toward a per se cure for the ailment.